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GENERAL ARTICLES

PULSUS PARADOXUS AND PLEURISY

By W. ERIC GIBB

From St. Bartholomew's Hospital

In 1888 Samuel West wrote: "Nearly all the instances recorded of pulsus paradoxus have occurred in connection with pericarditis or with affections of the mediastinum." Undoubtedly it is most commonly found in pericardial effusion and indurative mediastino-pericarditis, but it is noted also in the course of affections primarily of the lungs or pleuræ. Its occurrence in two cases of massive pleural effusion is here recorded, and the clinical significance briefly discussed.

1. The Occurrence of Pulsus Paradoxus in Cases of Mediastino-Pericarditis

In his Hunterian lecture, Holmes Sellors (1946) reminded us that it was Richard Lower (1669) who first recognised constriction of the heart by a thickened pericardium as a cause of cardiac failure and who first described an inspiratory suppression of the pulse in this condition. Later, Greisinger (1854) recorded that not only may the pericardium be found to be adherent and enormously thickened, but also the adjacent anterior mediastinum, forming with it a mass of indurated fibrous tissue. He observed an irregularity and frequently an intermittence of the pulse, "pulsus inspiratione intermittens," which became imperceptible with each inspiration; and also that the cervical veins, instead of collapsing, as is usual with free entry of blood to the right heart during expansion of the chest, distended with each inspiration. This he considered was due to the dense fibrous adhesions of the mediastinum dragging upon the innominate veins and the superior vena cava. Kussmaul (1873) described three similar cases and explained the diminution and cessation of the pulse as due to the stretching and narrowing of the aortic arch by the same adhesions. Similar cases of chronic hypertrophic pericarditis with the same intermission of the radial pulse and fullness of the neck veins during inspiration have been described by Traube, Baümler and other observers, both in England and Germany. Greisinger's original term "pulsus inspira-

tione intermittens " is an accurate and descriptive one. It implies a waning and waxing of the pulse amplitude with concomitant fluctuations in the systolic and pulse pressures, and it must assume that the inspiratory waning of the arterial pulsation should occur in all the peripheral arteries and therefore not be due to vascular compression at the axillary inlet as, for example, that of the subclavian artery between the first rib and the clavicle causing constriction of the blood flow to one or both arms (Falconer and Weddell, 1943; Learmonth, 1947; Telford and Mottershead, 1947); and also that there should be no marked irregularity of the action of the heart, as in sinus arrhythmia of a high order.

2. Its Occurrence in Pericardial Effusion

The presence of pulsus paradoxus may be the first indication of the existence of a pericardial effusion, and it occurs not infrequently with large pericardial effusions; but the mechanism of production appears to be different from that seen in chronic indurative mediastino-pericarditis and in other disorders of the respiratory and vascular systems.

Gauchat and Katz (1924) observed its presence when intrapericardial pressure was high, and its disappearance when this pressure was reduced by tapping; and they considered that inspiration reduced the pressure gradient which promotes flow of blood to the heart by reducing pressure in the great veins about to enter the pericardium further than in the intrapericardial portions of the vessels and in the auricles.

Fletcher (1945) demonstrated that paracentesis of a pericardial effusion resulted in a marked fall of the right auricular pressure, the filling pressure of the right auricle remaining unchanged.

3. Its Occurrence in Normal Individuals

Williams (1907) noted how frequently he was able to find respiratory undulations of the pulse wave occurring in normal boys entering Clifton College and was convinced that pulsus paradoxus was a pathological exaggeration of a physiological condition. And it is now well recognised that in normal persons, as well as in abnormal subjects, on deep inspiration the pulse beats may weaken, this being considered to be due to the blood being pooled within the pulmonary circulation as its capacity increases when the intrathoracic pressure falls.

Both Williams and Wenkebach (1910) pointed out the apparent relationship between pulsus paradoxus and intrapleural pressure variations: the former observer naturally stressing the effect of a markedly decreased pressure on the production of inspiratory intermission of the pulse.

4. Its Occurrence in Other Pathological Conditions

Since Kussmaul (1873) first described pulsus paradoxus occurring in chronic indurative mediastino-pericarditis, and Traube and Baümler noted its occurrence in massive pericardial effusions, it has been described in a number of other conditions. (Shreiber, Wenkebach, Roesler and others.)

In all, three clinical types were described by Wenkebach, which he termed:

(1) *The Mechanical Type.*—Due to mediastino-pericardial fibrosis in which the great vessels are further obstructed during inspiration. Reference has already been made to this.

(2) *The Extrathoracic Type.*—By compression of the subclavian artery (which can be artificially produced in some normal subjects by pulling the shoulder back and down, so bringing the clavicle closer to the first rib). This is not the true pulsus paradoxus, and was previously mentioned.

(3) *The Dynamic Type.*—Variations of pulse volume due to abnormal changes in intrathoracic pressure caused by the act of respiration. This type has been seen when respiratory embarrassment, due especially to thoracic disease, is present; and has been found in the presence of tracheal or bronchial stenosis, bronchial spasm, pneumonia, emphysema and bronchitis; and on occasions with pleurisy and also accompanying severe myocardial failure or following a severe hæmorrhage. It has also been found to occur immediately after pneumonectomy.

Roesler (1937) records details of two cases showing pulsus paradoxus; one of persistent bronchial asthma with emphysema (in which the intrathoracic pressure was augmented); the other, of chronic cor pulmonale with congenital cystic disease, in which gallop rhythm, cyanosis and clubbing also featured (the clinical picture being that of inadequate oxygenation of the blood). Here it is of interest to note the radiological findings and comments of Crowden and Harris (1929), who observed the changes taking place in the heart size and in the lung roots during the experiments of Müller and of Valsalva. In Müller's experiment (after expiration an attempted inspiration with a closed glottis) the heart, especially the right auricle, became larger and the lung roots increase in density, due to an increased flow to the heart being caused by a markedly negative intrapleural pressure; and the pulse disappeared, due to an increased lung reservoir, later reappearing if the experiment was prolonged. Crowden and Harris wrote: " The clinical picture corresponds closely to that seen in the attempt at inspiration against obstruction after a series of expiratory coughs in whooping cough and in some of the phases of the dyspnœa of asthma." The converse findings were seen in Valsalva's experiment (after inspiration a forced expiration against a closed glottis is attempted), the cervical veins distending and the heart diminishing in size, uncovering the lung roots; the increased (positive) intra-pleural pressure impeding the venous return, and the arterial pulse soon fading away although the heart continued to beat forcibly.

Böhme has also shown very beautifully the changes in heart size during such experiments on cats on ciné-radiographic films after injection of contrast media.

Rarely congenital abnormalities of the circulation, such as patent ductus arteriosus, have been reported as producing pulsus paradoxus (François-Franck; quoted by Reichmann, 1878). Cases such as these are of academic interest.

Gauchat and Katz noted one case of tuberculous pericarditis in which, a year later, a pleural effusion followed, and paracentesis of the pleural effusion did not relieve the pulsus paradoxus, as this was obviously due to the mediastino-pericarditis originating the year previously.

Case Records

CASE 1.—An airman, aged 18, was admitted to hospital on 10th July 1943 complaining of feverishness and pain in the left side of the chest which was pleuritic in character. He was of poor physique, had a temperature of 100°, a pulse rate of 108, and respiration rate of 22. A pleural rub was heard over the left chest anteriorly. Otherwise there were no abnormal clinical findings, but his evening temperature continued at 99°. There was no sputum and the blood count was normal. His sedimentation rate was 10 mm. in 1 hour. A chest X-ray was normal. Two weeks later, when he was transferred to an auxiliary hospital, there was still evidence of fibrinous pleurisy, and four days later he had a shivering attack, the temperature rising to 104° with recurrence of the pleural pain. He was then treated with sulphapyridine as he was thought to have pneumonia. As the fever did not settle he was then thought to have an empyema and was again transferred to hospital. On arrival he was found to be very ill, with a respiration rate of 30 per minute and signs of a large left-sided pleural effusion, which was mainly lymphocytic and was sterile on culture. Three days later the respiratory embarrassment had increased, the mediastinum was markedly displaced and the blood pressure was 90/50 mm. of mercury. Twenty ounces of fluid were removed with some relief, but two days later the mediastinum was even further displaced, the pulse was thready, the blood pressure was 80-90/50, and definite pulsus paradoxus was present. A further 30 ounces of fluid were aspirated, after which the mediastinal shift decreased and his condition improved, although there was still congestion of the right lung with audible rhonchi. There was never any evidence of pericarditis. Although he remained febrile for many weeks the effusion gradually subsided, leaving marked contraction of the left chest and pleural thickening. At no time did blood counts, sputum tests or examination of the pleural fluid suggest the effusion had been post-pneumonic.

CASE 2.—An officer was admitted to hospital in August 1942 with one week's history of right-sided pain of pleuritic type, fever and dyspnœa. He was found to have right-sided pleural effusion of moderate size and this was predominantly lymphocytic in character and sterile on culture. On complete rest his constitutional disturbance began to improve, but two weeks after admission there was a sudden exacerbation of fever with renewed pain and a rapid increase in the size of the effusion, which became massive and which was accompanied by considerable mediastinal shift. He had become dyspnœic at rest, somewhat cyanosed, and well-marked pulsus paradoxus developed, but there was no clinical evidence of pericarditis nor of change in the left cardiac contour radiologically. Thirty ounces of fluid were removed by paracentesis and following this the pulsus paradoxus vanished and the respiratory embarrassment was temporarily relieved. But pleural fluid continued to be exuded and signs of embarrassment and of pulsus paradoxus reappeared. His condition necessitated repeated withdrawals of moderate amounts of pleural fluid in the acute stage. Eventually the fever and the effusion subsided, considerable pleural thickening resulting.

Discussion

In those cases of serous pleurisy which are considered to be tuberculous in origin, the removal of large amounts of the exudate in the acute stage is not to be recommended. But in both these cases, one of massive right-sided effusion, the other of an equally large left-sided effusion, the degree of respiratory and

cardiovascular distress necessitated the removal of moderate amounts during the stage of exudation. In each case these procedures had the effect of reducing or abolishing the pulsus paradoxus, their immediate effect being to reduce the degree of mediastinal shift and also, presumably, the increased intrapleural pressure to some extent. In both cases there was undoubtedly a very complete collapse of the underlying lung and therefore a reduction to a minimum of the circulation through the lobes of this lung.

In discussing the reason for the presence of pulsus paradoxus in these two recorded cases the following points deserve attention:

(*a*) In neither case was there evidence of a pericardial effusion as a possible cause.

(*b*) While it is possible that it was associated with an interference with the venous return to the heart, and a large right-sided pleural effusion may do this by compressing the venæ cavæ, yet it is less likely that a left-sided effusion could do this unless the mediastinal shift has the effect of stretching and distorting the venæ cavæ. In this connection it is well known that sudden collapse and death occasionally occurs in cases of massive pleural effusion and it would be instructive to know whether a right-sided effusion is more liable to be associated with such a catastrophe than is a left-sided effusion.

(*c*) In each case there was good reason to believe that the massive effusion occurred in a pleural cavity with few, if any, adhesions and with a predominantly healthy underlying lung, and thus the increased intrapleural pressure caused collapse of the lung. In this way virtual cessation of one-half of the pulmonary circulation would result not only in an increased pressure in the pulmonary artery and an overfilling of the right side of the heart, but in a diminished return to the left side of the heart.

Summary

1. It is well known that " idiopathic " pericarditis and pleurisy may coexist; and that, when of tuberculous etiology, either may be fibrinous or exudative in character. But it is also well to recognise that pulsus paradoxus can occur in the presence of a large pleural effusion alone; and therefore, that when such an inspiratory waning of the pulse is present it must not be concluded that a pericardial effusion is also present. Whereas a pericardial effusion which gives rise to a pulsus paradoxus should give other signs of its presence, but frequently results only in moderate dyspnœa, a pleural effusion which is of sufficient size to give rise to pulsus paradoxus will also be accompanied by marked dyspnœa and distress. Its presence in a case of pleural effusion is certainly an indication for the removal of 20-30 ounces of fluid immediately.

2. Whereas the presence of pulsus paradoxus in cases of pericardial effusion and of mediastino-pericarditis is coincident with a severe interference with the venous filling of the right side of the heart, its presence in deep inspiratory breathing in normal subjects is associated with a pooling of blood within the pulmonary circulation and therefore a lessened venous return to the left side of the heart. In gross intrathoracic disease, including massive effusions and after pneumonectomy, there is likewise a decreased blood flow

to the left auricle, and therefore a lessened output from the left side of the heart.

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TUBERCULOMA OF THE LUNG MASQUERADING AS ENCYSTED PLEURAL EFFUSION

BY N. WYNN-WILLIAMS

From the Bedford Chest Clinic

THIS case is reported because of its radiological appearance, not because of its rarity. No one shown these films diagnosed the condition correctly, and doubt was not thrown on the diagnosis until the density of the central part of the shadow began to alter, suggesting the possibility of cavitation. The patient, a married woman of 41, was first seen on 25.8.47, when she complained of pain in the right shoulder and across the right upper chest anteriorly. It had been present for five days. Described as severe and made worse by deep inspiration, the pain was accompanied by cough and temperature up to 101°F. She had also suffered from lassitude for some weeks. There had been no sputum or hæmoptysis. The family and previous history was not relevant except that several months previously she had noticed a pain in her right chest on stretching upwards. This was diagnosed as muscular, but had been present on and off until the present complaint.

On physical examination she was thin and pale, weighed 7 st. (highest known 7 st. 4½ lbs.), and was 5 ft. 1 in. in height, temperature 98°, pulse 84. Over the right upper zone, anteriorly and posteriorly, the percussion note was impaired, expiration was prolonged, and a few fine râles were present, both before and after cough. Over the lower zone posteriorly breath sounds were slightly weak. No other abnormality was noted in any of her systems.

Her Erythrocyte Sedimentation Rate was 9/200 mm. at one hour (Westergren). A complete blood count on 29.8.47. was within normal limits. X-ray report 25.8.47. P.A. film (Fig. 1)—Heart and mediastinum normal. Left

costo-phrenic angle diminished. *Right lung.*—There is a circular homogeneous opacity in the region of the interlobar septum. This appears to extend along the interlobar fissure laterally, and is continuous with the hilar shadow medially. Right lateral film (Fig. 3)—There is a roughly circular opacity anterior to the hilum. This has a prolongation that runs forward in the direction of the lesser fissure, is ribbon-shaped, and curves slightly downwards.

A provisional diagnosis of encysted effusion of the lesser fissure was made.

In view of the small size of the presumed effusion and its distance from the anterior chest wall, it was not considered justifiable to try and obtain a specimen of fluid. The patient was put on bed rest at home under excellent conditions, and a near sanatorium régime maintained. Further films were taken at monthly intervals. After three months no change could be observed in the shadow except that its extension forward on the lateral film was slightly smaller. The patient appeared well and was afebrile. She did, however, complain of indefinite pains across the right side of the chest anteriorly. At this stage she was allowed out of bed, and gradually permitted to increase her activities. She was never placed on exercise. During the next three months she complained of lassitude, but otherwise appeared well. Monthly radiographs were continued, and these showed no change until 14.1.48, when the shadow began to change very slightly in density towards its centre. By 10.3.48, the appearance of the P.A. film had definitely changed, suggesting the possibility of cavitation (Fig. 2). This was not obvious in the lateral film, but was confirmed by tomography (Fig. 4), a cut at 12 cm. showing this to the best advantage. The possibility of this being fibrin laid down round the edge of the effusion was considered. However, it was decided that further investigation was justified, and on 28.4.48, the patient was admitted to the Brompton Hospital, under the care of Dr. F. P. Lee Lander.

The following investigations were carried out. Erythrocyte Sedimentation Rate 8 mm. at one hour. Blood Count Hb. 93 per cent. Red cells and white cells within normal limits. Six sputa examinations were negative for tubercle bacilli, direct and on concentration.

12.5.48 Bronchoscopy (Mr. Price Thomas). Pus seen issuing from right upper lobe bronchus. It appeared to be drawn down, and the segmental orifices could be seen through direct vision telescope. Nil else abnormal seen.

18.5.48. Bronchogram. The anterior division of the pectoral bronchus of the right upper lobe is dilated and blocked. Lipiodol did not run into the anterior part of the pectoral segment. Fig. 5 (right lateral) shows the blocked division ending at the abnormal shadow.

A diagnosis of tuberculoma of the pectoral segment of the right upper lobe was made, though the possibility of a new growth was borne in mind. Thoracotomy was not advised.

The patient returned home and complained of sputum for the first time, following these investigations. This proved positive for tubercle bacilli by fluorescent microscopy on 21.6.48. During early June she had complained generally of not feeling well, and started to run a temperature during the second week of the month. This rose to 102°F. on 18.6.48, and within a few days abnormal signs appeared in the right lung. By 26.6.48 there was impairment of percussion note, bronchial breathing, and many ante- and post-tussive

râles from the second rib down and into the axilla anteriorly. The same signs were present over the upper two-thirds of the lung posteriorly. A radiograph on this date showed mottling over the upper zone, heavy shadowing over the mid-zone, and mottling over the lower zone. A lateral film showed heavy shadowing over the whole sub-apical (postero-lateral) segment and over the axillary part of the pectoral segment of the right upper lobe, suggestive of collapse. The left lung remained normal.

The sputum by this time was positive for tubercle bacilli on direct smear. Owing to the residual lipiodol that was present in the right lung, it was, apart from the segmental lesions, impossible to be sure of the extent of the disease there; a point that should be noted, as bronchoscopy and bronchography were discussed and not considered justifiable as early as December 1947. It is also of interest that the first symptoms of active disease occurred so soon after these manipulations.

The question as to whether the performance of a bronchogram is likely to be dangerous in pulmonary tuberculosis has often been discussed. Murphy (1934), who did bronchography in 99 cases of pulmonary tuberculosis, noted no evidence of spread of disease. Mandelbaum also noted no complications following 250 lipiodol injections in 90 patients. Dormer *et al.* (1945) report that they have done over 2,000 bronchograms in pulmonary tuberculosis of all types. They state that they have never seen a case the worse for instillation of lipiodol.

The further history of this patient is not really relevant to the discussion, but a right A.P., followed by adhesion section, has obtained satisfactory relaxation of the right lung, and she appears to be on the road to recovery.

Discussion

Tuberculoma of the lung is also described as caseous nodular tuberculosis or as a solitary round focus. There is remarkably little in the literature on the subject, in view of the common occurrence of the condition. It is suggested that a tuberculoma may arise either by inhalation, or that it may be hæmatogenous in origin. A tuberculoma is roughly spherical in shape, of fairly uniform density and with sharply defined borders. In size it may vary from about 1 to 5 cm. Pathologically, it consists of round structureless, caseous masses, with a variable amount of calcification. It is akin to a caseous pneumonic area. Fruchter (1935), considers that a tuberculoma represents the development of exudative focal tuberculous inflammation in an unusual form. Pierson (1942) looks upon a tuberculoma as a circumscribed mass of tuberculous granulation tissue without surrounding inflammation. Brock (1946), demonstrates a tuberculoma that he considers to be of inhalation origin and enclosed in a dilatation of the bronchus itself. Kourilsky *et al.* (1948) take a similar view to Pierson.

It would appear from these papers that the solitary round focus may be regarded as a tuberculous focus, unusual only in that it is circumscribed and not accompanied by any surrounding inflammation. It is surrounded by a fibrous capsule. Like other tuberculous foci, it may caseate and finally form pus, or proceed to calcification. Most commonly the fibro-caseous wall

PLATE I.

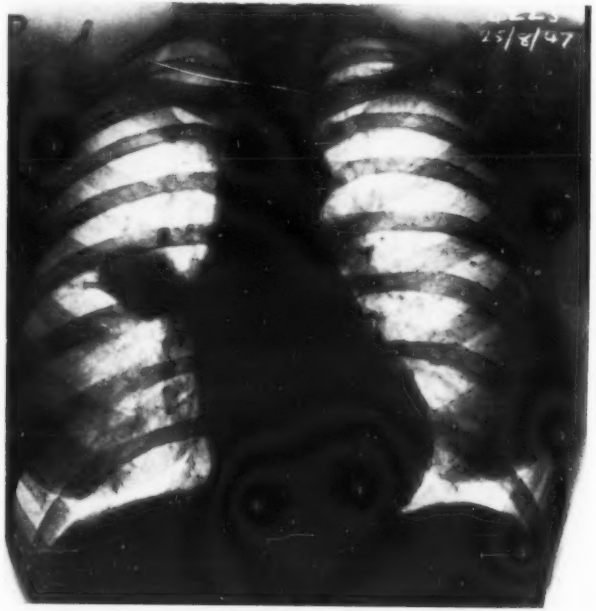

FIG. 1.

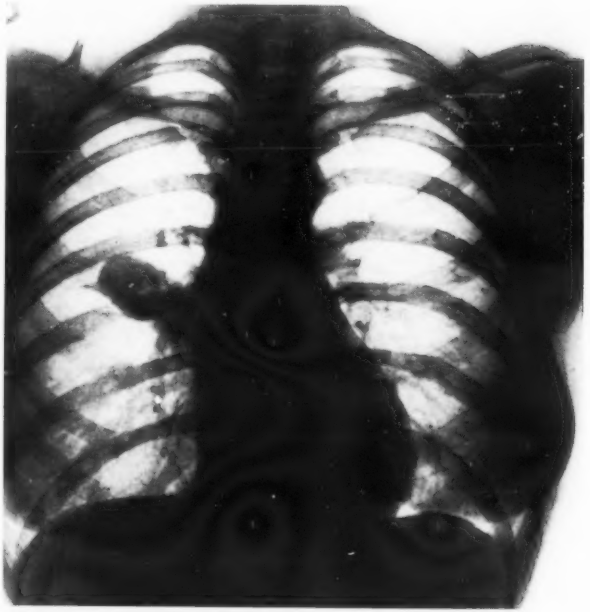

FIG. 2.

To face p. 1.

PLATE II.

FIG. 3.

FIG. 4.

FIG. 5.

To face p. 9.

thickens greatly, some calcium is deposited, but centrally there remains a focus of more or less activity, which is a potential danger to the patient.

A condition that simulates tuberculoma, both clinically and radiologically, is an isolated cavity with a blocked bronchus. If the cavity becomes filled with inspissated caseous material, a rounded circumscribed shadow is formed that can only be distinguished from a tuberculoma if the series of events has been followed radiologically.

The particular interest of this case depends on the difficulty that has always been experienced in the differential diagnosis between interlobar effusions and pulmonary lesions abutting on the fissures. A review of the literature over the last twenty years shows very clearly how opinion has altered regarding the radiological diagnosis of interlobar fluid. Physical signs have never been of much value, and the conception of its being a frequent occurrence arose as it became possible to take chest films of reasonable definition, and shadows in the region of the fissures were observed. Many shadows that were once diagnosed as interlobar fluid are now known to be caused by segmental lesions of lung.

An encysted interlobar effusion is usually described as being elliptical or spindle-shaped. It tends to become broader and rounder as the tension within increases until a circular shape may be assumed. The borders should be sharply defined from the lung, unless there is an associated lesion of the lung, when only one border would be sharply defined and curved. It should be along the line of a fissure, and small peaks or extensions along the fissure, or thickening of the interlobar pleura along the fissure, are often present. Watched over a period, the shadow may be seen to extend along the fissure.

An interlobar effusion communicating with the general pleural cavity is much more common, and is really the extension of a general effusion into one of the interlobes. These effusions tend to be more triangular in shape with the base of the triangle pointed towards the general pleural cavity, the apex along the fissure. The sides of the triangle, however, are often rounded. These triangular shaped effusions are those confused with segmental lesions of the lung. The rounded encysted effusions are those with which we are concerned, and those that may imitate any rounded shadow in the lung itself.

In the case under review, Fig. 1 would appear to conform strictly to the appearances of an encysted effusion of the lesser fissure, lying over the line of the fissure, and with a peaked extension along the fissure ending in thickened interlobar pleura. The shadow, however, is continuous on its inner side with the hilum. It has been stated that an interlobar effusion is always separated from the hilum by a band of air containing lung. However, though this is certainly more common, many authorities state that this is not a necessary condition (Fleischner, 1926). It depends on the depth of this interlobar fissure (Shanks, Kerley and Twining, 1938), and as this rarely reaches the mediastinal surface of the lung, an effusion is more common in the outer lung field. Examples of round effusions of the lesser fissure are shown by the above authors.

Fig. 2, lateral view, shows a rounded swelling in the position of the interlobe. The ribbon-like shadow extending forward and curving downwards ought perhaps to have thrown doubt upon the diagnosis, as it is not shaped in the usual way.

Freedman (1931) describes a case of great interest in relation to this shadow. A female of 39 years of age, complaining of hæmoptysis, raised temperature, etc., showed on a P.A. film a triangular shadow arising from the right hilum. On the lateral film a ribbon-shaped projection forward with the same slight curve downwards, as in our case, is shown. This, today, would undoubtedly be diagnosed as collapse of the anterior division of the pectoral segment of the right upper lobe, though Freedman describes it as an effusion of the lesser fissure.

It seems likely that this ribbon-like curve was caused by extension of the tuberculoma into the lower border of the upper lobe, and by this border overlapping the middle lobe.

Encysted interlobar effusion is taken first in the differential diagnosis of this shadow, as this was the mistaken diagnosis made. Encysted effusion can be caused by a tuberculous pleuritis. This could be clear or purulent. It could also be caused by pyococcal organisms, when it would be commonly described as an empyema. Encysted empyema is rare in the absence of inflammation elsewhere in the pleural cavity. When occurring alone, it is most frequently associated with lung abscess, and according to Copleman and Neuhof (1941), it is always due to a lung abscess perforating into the interlobe. Severe constitutional symptoms, or at least the history of these, should be present. A polymorpholeukocytosis should also be present. Encysted fluid can also occur in heart failure.

A rounded shadow may also be caused by chronic pulmonary abscess of long standing. Such abscesses may be calcified, and yet careful search may show no evidence of tuberculosis. Graham and Singer (1936) reported three such cases.

Primary or secondary carcinoma of the lung would probably be the next most likely diagnosis in a shadow of this type. Primary carcinoma usually shows some variation in the density of the shadow, although this is slight. The rare sarcoma or chorion-epithelioma of lung show no variation in density. The latter can usually be confirmed by a positive Aschheim-Zondek test. Secondary carcinoma may also show a single rounded shadow in the lung, though usually other nodules occur before long. Secondaries from the kidney (Hypernephroma) or testis (teratoma) are the most likely primary sites of an isolated secondary nodule. They are frequently associated with hæmoptysis.

Benign tumours are rare causes of such a shadow. Adenoma of the bronchus by growing outwards from the bronchus and not occluding its lumen can cause such a shadow. It is associated with repeated hæmoptysis. Chondroma and lipoma, being composed of cartilage or fat, are very radio-translucent, and so cause faint shadows, which should not be mistaken.

A dermoid cyst might be thought of, but usually, though spreading into the lung field, a dermoid has a broad base on the mediastinum, and on the lateral view is usually higher in the chest and in the anterior mediastinum. A dermoid is also seldom homogeneous with varying penetration.

Cysts of lung might possible give such an appearance. An air cyst filled with fluid shows a round shadow, sharply demarcated from lung and quite homogeneous. An air cyst filled with blood would usually give a history of hæmoptysis. An echinococcus cyst may have the same characters of sharp

definition. It is apt to be rather less strictly rounded, especially in the P.A. view. There may be a shell of calcium in the adventitia of the cyst. Occasionally air may penetrate between the adventitia and the laminated membrane, giving the appearance of a crescent. This perivesicular pneumocyst is said to be pathognomonic of hydatid cyst (Barrett and Thomas, 1944).

Enlarged intra-bronchial lymph nodes from Hodgkin's disease or lymphosarcoma can show a rounded shadow in the lung with no obvious connection with the mediastinum. The latter, however, is usually increased in breadth.

An unusual condition that might give such a shadow is a hæmangioma of the lung (arterio-venous aneurysm). Barnes *et al.* (1948) state that this may appear as a circumscribed opacity usually connected to the hilum by a shadow which represents the vessels running to and from the tumour. It may sometimes be seen to pulsate on the screen. Tomography is of value in showing up the vessels. Clinically, the patient is usually cyanosed.

Finally, there are inflammatory conditions of the lung, such as the mycoses, which usually show diffuse shadows, but may, as in tubercle, present as rounded isolated lesions. Gumma is said also to produce such an appearance.

Summary

(1) Interlobar effusions, particularly those of the lesser fissure, are discussed.

(2) A segmental lesion of the lung may easily be mistaken for an interlobar effusion. This is well realised in relation to the greater fissure, but less so with regard to the lesser fissure.

(3) Clear-cut rounded margins to the shadow are no guarantee of an interlobar effusion.

(4) Bronchography and bronchoscopy are valuable aids in establishing the diagnosis.

(5) A review of the literature makes it obvious that encysted interlobar effusion is diagnosed with much less frequency than formerly.

(6) Particular caution should be used in diagnosing encapsulated fluid in the absence of fluid in the general cavity.

My thanks are due to Dr. F. P. Lee Lander for his help in investigating this case, and to Dr. L. G. Blair, for permission to reproduce the bronchogram shown in Fig. 5.

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A KEY TO THE CLASSIFICATION OF PULMONARY TUBERCULOSIS

By MILOSH SEKULICH*

The many different clinical forms of pulmonary tuberculosis, as well as the different names under which they may be described, tend to obscure the clinical picture as a whole. The confusion has largely arisen as a result of the slow development of our knowledge of the problem. Classification is still far from complete, and different authors use different systems, according to their own experience and practice.

A simple, correct and practicable classification of tuberculosis is necessary, not only for mutual understanding within the medical profession, but also for the preparation and examination of statistical records. A recent attempt at such a classification, by Prof. J. E. Kayser-Petersen of Jena, covering pulmonary as well as non-pulmonary tuberculosis, involved no less than fifty-five different forms (" Der Tuberkulosearzt," 1948, Georg Thieme, Stuttgart).

In any case, a basis must be found for the classification of tuberculosis which would include such opposites as " open " and " closed," acute and chronic, active and inactive, regressive and progressive, unilateral and bilateral, destructive and non-destructive, cavernous and non-cavernous, etc.

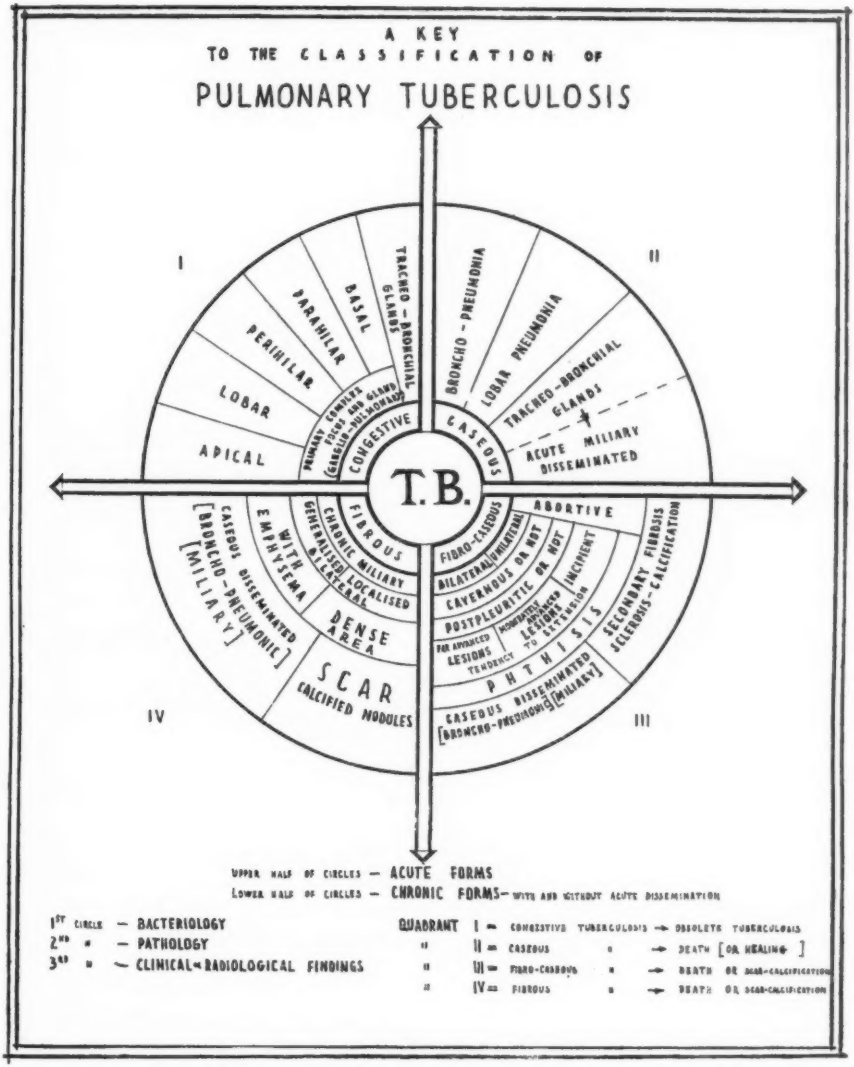
Suggested Classification

The classification suggested here is based on bacteriology, pathogenesis, pathology, clinical and radiological findings. It is the result of many years' experience, and was found to be capable of application to some two thousand cases, the records of which are unfortunately not now available. Some of these were under observation for fifteen to twenty years. In some hundreds of cases the correctness of the classification was verified by autopsy or by cure over a period of more than five years. The two important points of this classification are: (1) that every form of the disease can be visualised in continuity from beginning to end; and (2) that the clinical picture corresponds with the pathological process involved. The advantage of the classification is that when a diagnosis is made, it not only determines the existing condition, but also the history of the process and possible future developments. It can be understood by any doctor in any country. This unifying conception allows of discarding many different names for the same thing.

The classification is depicted graphically in the accompanying diagram, which consists of three circles with one common centre and four quadrants (see Diagram). The first circle represents bacteriology; the second, pathology, the third, clinical and radiological findings. By horizontal and vertical diameters we obtain four quadrants, each of which contains a different group of forms of the disease. The two upper quadrants contain all acute forms, and the two lower contain all chronic forms with or without acute dissemination.

* Formerly in charge of the Clinic for Internal Diseases at the Municipal Hospital, Belgrade.

The pathology of any form of pulmonary tuberculosis may include several elements—*e.g.*, congestion, infiltration, caseation, fibrosis, sclerosis, and combinations of these. Four of these pathological elements can be selected as the dominating character which determines the clinical picture of the four



groups. Thus, in the first group the *congestive* element is the most important, and this is therefore the group of CONGESTIVE FORMS. The second group, determined by the *caseous* element in the pathology, includes all the CASEOUS FORMS. In the third group the *fibro-caseous* element is the distinctive one, and

it therefore includes all FIBRO-CASEOUS FORMS. Similarly, in the fourth group the *fibrous* element is dominant, and this group includes the FIBROUS FORMS.

Every form of the disease is at some time associated with activity. In this classification it is understood that the activity of congestive forms gradually subsides and disappears completely; that of caseous forms increases until death (except cases of miliary tuberculosis cured by streptomycin); that of fibro-caseous and fibrous forms fluctuates with periods of quiescence and periods of activity, the latter being associated with new areas of congestion and caseation.

Again, in every case, the question of dissemination may arise at any stage. This may lead to the spread of the disease in another part of the lung or to generalisation in other organs. If the lungs are involved by dissemination from a lesion in another organ—*e.g.*, bones or joints—we either classify the case as surgical tuberculosis, excluding it from our classification, because the pulmonary lesions are then terminal, or include it among the caseous forms.

I. CONGESTIVE FORMS

Congestive forms begin as a benign primary complex: there is a tuberculous primary focus and a tuberculous regional gland. Millions of people have recovered from this first infection without ever realising that they have been ill. The primary focus in this case is followed by an insignificant perifocal reaction without giving any clinical signs or symptoms. A minority of cases, on the other hand, give a perifocal reaction sufficient to produce a definite clinical picture and radiological findings. These are acute forms, and are therefore associated with the symptoms and signs of activity. They are revealed radiologically as apical, lobar, perihilar, parahilar and basal infiltrations. They are called by some authors " epituberculosis." Their activity lasts from a few weeks to a few months and ends in the majority of cases with healing by complete resorption. In a minority, however, a residua remains of obsolete tuberculosis, that is, scarring or calcification. This is shown by radiography during life or, in case of death from intercurrent disease, at autopsy.

It is clear that a diagnosis of congestive tuberculosis refers to an acute, benign and curable form (with the bacillus occasionally found in sputum or stomach contents), ending either in complete resorption or in an obsolete healed lesion. In both cases tuberculous latent infection usually persists for life, this being expressed by a positive tuberculin reaction. While millions live without incurring new tuberculous disease, a small minority do so and, being allergic from their primary infection, develop chronic lesions. A healed primary complex is therefore equivalent to vaccination against tuberculosis, and is an effective protection for the majority of the population.

II. CASEOUS FORMS

Caseous forms begin as a primary complex. Instead of a perifocal congestive reaction, however, a dissemination of caseous lesions takes place by air, lymphatic or blood routes, with ultimate formation of caseous bronchopneumonia, caseous pneumonia, miliary tuberculosis or mixtures of these. Not

infrequently, other organs are involved in the dissemination, especially the meninges. The clinical picture corresponds exactly with the pathological changes, the caseous element being the key lesion.

Rarely a caseous form may develop from a partly calcified primary complex.

III. Fibro-Caseous Forms

Fibro-caseous forms develop in an organism with a "healed" primary lesion—that is, in a tuberculin-positive person. They develop after a variable period of years, or even without any interval at all. A chronic form with a long course is characteristic of this group. The basic pathology is the combination of fibrous and caseous elements interfering with each other. The degree of fibrosis developed is proportional to the resistance of the organism.

If there is a "rudimentary" lesion without any activity or tendency to dissemination, giving hæmoptysis as the only sign, we have an abortive form. The majority of fibro-caseous forms begin with unilateral lesions, rarely bilateral. This "fruhinfiltrat," or Assman's focus, may become arrested, but sooner or later, as a rule, it requires collapse therapy. Without treatment, the initial form gradually passes to the "moderately advanced" and "far advanced" stages. In fact, those three stages correspond to the three stages of the Gerhard and Turban classification. During the course of all three stages a sudden caseous dissemination can take place and aggravate the course, often fatally. The initial stage can, without collapse therapy, sometimes pass to a secondary fibrous stage of scarring and calcification. The moderately advanced stage can become fibrous only under prolonged collapse therapy. The far advanced stage is incurable. Fibro-caseous forms can be unilateral or bilateral, and with or without cavity formation or pleural involvement. They have a tendency to spread, and the appearance of the patient is phthisical, except in the initial or secondary fibrous form. Caseous dissemination can occur at any stage or in any fibro-caseous form, often with fatal results.

IV. Fibrous Forms

Fibrous forms are similar in nature to fibro-caseous forms, except that they are characterised by an extreme fibrosis, and by a caseous element of very short duration. They develop on both sides simultaneously, beginning as small nodules with a strong fibrotic tendency. Occasionally this process is repeated many times, until the lungs appear to be "peppered" with fibrous nodules, thus producing the form "chronic miliary tuberculosis." These nodules may have a surrounding area of congestion or be without it (*granulie froide*). Their evolution is usually in the direction of dense or diffuse fibrosis, with accompanying emphysema. But there are some cases, relatively rare, in which caseous dissemination occurs just as in the fibro-caseous forms, thus interrupting the very slow chronic course characteristic of the fibrous forms. Indeed, this kind of tuberculosis is the most prolonged of all. It may last for many decades, sometimes in an active phase, "open," and dangerous, particularly to children, and at other times "closed" and quiescent.

A Key to the Classification of Pulmonary Tuberculosis

I. The Congestive Forms

Benign Ghon Complex.

Apical, Lobar, Perihilar, Parahilar, Basal — congestive tuberculosis (epituberculosis).

Obsolete tuberculosis (Calcified Ghon complex).

II. The Caseous Forms

Malignant Ghon complex.
Caseous tracheo-bronchial glands.
Acute miliary (disseminated) tuberculosis.
Caseous bronchopneumonia.
Caseous lobar pneumonia.

III. The Fibro-Caseous Forms

Abortive form.

Initial, Moderately advanced, Far advanced — fibro-caseous spreading phthisis.

unilateral cavernous postpleuritic — or bilateral or non-cavernous or non-pleuritic

Initial, Moderately advanced, Far advanced — caseous disseminated fibro-caseous phthisis.

Fibro-caseous secondary fibrous tuberculosis.
Sclerosis.
Calcified nodules (Calcaria).
Fibrothorax.

IV. The Fibrous Forms

Localised–Generalised — chronic miliary tuberculosis (*granulie froide*).

Dense, Diffuse — fibrous tuberculosis with emphysema.

Caseous disseminated fibrous tuberculosis.

Localised, Generalised — calcified nodules.

Sclerosis — Calcaria.

By the courtesy of Prof. J. H. Dible, I have been privileged to study the records of 4,000 consecutive necropsies at the British Post-Graduate Medical School, Hammersmith. These records were so complete as regards clinical, bacteriological and biochemical findings, morbid anatomy and histology, that they formed ideal material for testing the value of the classification suggested above. It was found, in fact, that each case with tuberculous lesions fitted perfectly into each appropriate place in the accompanying diagram.

Of the 4,000 necropsies, 286 showed tuberculous lesions, and these were classified without difficulty, according to the scheme as follows:

I. Congestive forms, all obsolete	30
II. Caseous forms	36
III. Fibro-caseous forms	132
IV. Fibrous forms	53
Total pulmonary tuberculosis	251
Surgical tuberculosis	35
Total	286

Detailed classification within each main group of the 251 examples of pulmonary tuberculosis is shown in the following table:

I. Obsolete tuberculosis:	
Calcified Ghon complex	11 cases.
„ „ focus	12 „
„ gland root lung	1 „
„ „ mesentery	6 „
Total	30 „
II. Acute miliary tuberculosis (with Ghon focus—9 cases)	21 cases.
Caseous bronchopneumonia (with Ghon complex—7 cases)	15 „
Total	36 „
III. Fibro-caseous phthisis:	
Initial	50 cases.
Moderately advanced	4 „
Far advanced	6 „
Caseous disseminated:	
Initial	30 „
Moderately advanced	15 „
Secondary fibrous tuberculosis	5 „
Fibro-caseous secondary fibrous tuberculosis	22 „
Total	132 „
IV. Fibrous forms:	
Localised chronic miliary tuberculosis	5 cases.
Dense fibrous tub. with emphysema	5 „
Diffuse fibrous tub. with emphysema	12 „
Caseous disseminated fibrous tuberculosis	4 „
Sclerosis	27 „
Total	53 „
Total	251 cases.

It seems that the method of classification suggested in the paper now presented provides a key to the practical classification of pulmonary tuberculosis, scientifically correct, easily understandable, and readily applicable for statistical purposes.

The author would like to record his gratitude to Prof. J. H. Dible, who permitted him to use records from his Department; also to Dr. H. Stanley Banks, who kindly helped in editing the English phraseology of this paper.

PNEUMOMEDIASTINUM COMPLICATING ARTIFICIAL PNEUMOPERITONEUM

By JOHN B. MORRISON

Although pneumomediastinum is a not uncommon complication of artificial pneumoperitoneum, the reason for reporting the following case is that the symptoms and signs of the complication suggested that the air might have been within the pericardial sac.

The patient, a man of twenty-two, was admitted to hospital suffering from pulmonary tuberculosis affecting the lower lobe of each lung. In the right lower lobe there was a cavity of 3·5 cm. diameter (Plate III, Fig. 1). His sputum was positive for *M. tuberculosis* on direct smear.

It was decided that a right phrenic crush followed by pneumoperitoneum should be the treatment of choice. After the right phrenic crush, the right dome of the diaphragm rose 1½ ins. and was immobile. Seven days later the pneumoperitoneum was induced, 700 c.c. of air being injected into the right iliac fossa. As the air flowed in the patient had some discomfort in the epigastrium, and towards the end of the induction he was nauseated and faint and felt pain under the left costal margin and in the right shoulder region. Both pains lasted only ten minutes. Some thirty minutes later he was allowed to sit upright. Three hours later he had a very sharp pain over the precordium, not aggravated by deep breathing or coughing at first. It was severe enough to require Alopon ⅙ grain. Pain was felt over the precordial area on coughing and on deep breathing on the following day. By the third day after the induction the pain had gone. During the attack of severe pain the patient was extremely distressed but not breathless. He had a small area of subcutaneous emphysema between the fourth and sixth ribs anteriorly, but no superficial air was detected elsewhere in the neck, thorax or abdomen. The apex beat was not visible or palpable, the area of cardiac dullness was diminished, and on auscultation a crackling sound was heard over the precordium with maximum intensity at the cardiac impulse. It accompanied the heart beat and was unaffected by holding the breath. The adventitious sound resembled pericardial friction, and no better description can be found for it than that of Stern, quoted by Graebner (1939), who found that air within the mediastinum produced a crackling murmur at the left border of the sternum, best heard on expiration, and resembling the crushing of cellophane in the hand. A radiogram taken the day after the induction showed a distinct line just outside the left cardiac border, an area of translucency intervening between the line and the cardiac margin. All clinical signs disappeared by the third day following the induction and no further air was introduced. A right-sided artificial pneumothorax was done later and the cavity closed.

Discussion

Graebner (1939) notes that in pneumomediastinum complicating acute obstructive laryngitis a characteristic crackling murmur can be heard over the precordial area. In two cases of croup treated by tracheotomy a similar murmur was heard and on radiological examination air was found within the pericardial

PLATE III.

FIG. 1.

To face p. 18.

sac. This air produced a characteristic linear shadow parallel with the left cardiac border, an appearance similar to that noted by the writer. Banyai and Jurgens (1939), however, in a paper on mediastinal emphysema complicating artificial pneumoperitoneum, reproduce two X-rays showing air lying along the left border of the heart. Hamman (1945) also states that in mediastinal emphysema a sharply defined band outlining the course of the pericardium, especially along the upper part of the left border, can be seen; and Schwarz, McIlroy and Warren (1946) describe a thin line of air parallel to the cardiac border on each side.

The presence of retrosternal pain, diminution in the area of cardiac dullness and the crackling murmur described, in the absence of other symptoms or signs of mediastinal emphysema such as a choking sensation, pain on swallowing and emphysema of the neck, suggested the possibility of air having entered the pericardial sac by some defect. At first it was thought that the radiogram confirmed this, but Dr. P. Kerley pointed out that the top of the abnormal line to the left of the heart lay at least 1 in. above the pericardial insertion, thus disproving the diagnosis of pneumopericardium. He also suggested that a mediastinal pleurisy might have prevented air from reaching the tissues of the neck.

Summary

An example is described of pneumomediastinum complicating induction of artificial pneumoperitoneum. Precordial pain, diminished cardiac dullness and a crackling murmur accompanying the heart beat were the presenting features. The difficulty in distinguishing this condition from pneumopericardium on radiographic examination is noted.

I wish to acknowledge my indebtedness to Dr. P. Kerley, for interpreting the radiogram, and to thank Dr. M. A. Foulis, Medical Superintendent, Robroyston Hospital and Sanatorium, Glasgow, for access to the case record.

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REVIEWS OF BOOKS

Procedure in Examination of the Lungs. By ARTHUR F. KRAETZER, M.D. Revised by JACOB SEGAL, M.D., F.A.C.P., F.C.C.P. Pp. 147. Oxford University Press: Geoffrey Cumberlege. Price 18s.

A striking feature of this useful little book is the true appreciation of the needs of the humble beginner for whom it is intended. The time-honoured procedures of inspection, palpation, percussion and auscultation are retained in order to ensure the development of method. The student is encouraged to interpret physical signs in terms of pathological processes, and much mystery is thereby dispelled. He is not given a list of particular diseases and the signs he might be expected to find in them, nor is he burdened with the more rarely elicited signs and the names of the physicians who described them. In-

stead, he is taught how to elicit the common physical signs, progressively to assimilate the facts and eventually to reach an inductive conclusion. A simple and acceptable order is brought out of the chaos of râles, and their possible causation and significance clearly explained. The appendix contains notes on essential investigations and illustrative completed case histories. The addition of a short explanation of the diagrammatic representations of physical signs in common use would be an advantage. It is a book for the student about to enter the medical wards, and he will find it both readable and helpful.

F. H. S.

Surgical Extrapleural Pneumothorax. By DONATO G. ALARCON. Imprenta Universitaria Mexico, 1948. Price not stated.

Extrapleural pneumothorax reached its greatest popularity in the period 1937-39, but thereafter fell from favour because of its high mortality and frequent complications. A few thoracic surgeons persevered in its use and by careful selection of cases and close attention to operative and post-operative detail achieved much more favourable results. The author was one of these few, and in this book he sets out to justify the use of the operation in the light of his own experience and results.

A short historical sketch is followed by a lengthy discussion of the indications for the operation. The most suitable cases in the author's experience are those with unilateral lesions, not more than one year old and with a cavity not more than 4 cm. in diameter. In describing the operative technique the danger of cutting through adhesions in the extrapleural plane is stressed; when such resistance is met the periosteum and most of the intercostal bundle are if necessary included in the strip. All cases are drained by a thin, clipped-off rubber catheter which is removed in 24-48 hours, after running off any fluid which has collected; if the space has become infected the catheter is retained for irrigation purposes.

Early conversion to Oleothorax is favoured, especially when attendance for refills is difficult or when undue obliteration is occurring. Oleothorax is also advocated in the treatment of tuberculous infection of the extrapleural space.

The author's results are unfortunately given in such a manner that they lose much of the conviction which they might have had. The only criterion of success is sputum conversion and no mention is made of the length of follow-up, fitness for work or relapse rates. The operative mortality of 277 operations over a period of eight years was 4·5%, but this figure is somewhat discounted by the fact that 6% of the cases have been " ignored " for no stated reason. The book is written in a style which is frequently difficult and at times impossible to understand, and is full of medical jargon. No apparent attempt at editing the book has been made, printer's errors abound and spelling mistakes are legion. No proper reference in the text is made to any of the 127 X-ray photographs, and finally there is no index. These defects are not worthy of an elaborate monograph.

A. T. M. R.

" *Irregular Discharge* ": *The Problem of Hospitalization of the Tuberculous.* By WILLIAM B. TOLLEN, Ph.D. 1948. Pp. 64. Veterans' Administration, Washington, 25, D.C. Price 20 cents.

During the year July 1946 to June 1947, 54 per cent. of the discharges from V.A. tuberculous hospitals were " irregular "—*i.e.*, a result of the patient's own actions and not with medical approval. This report condenses the results

of a tremendous amount of team-work by social workers engaged in an exhaustive enquiry into the causes of this major problem, which is shown to be not confined to V.A. hospitals, but to be almost a characteristic of the tuberculous patient under treatment. The basic reasons are found to be domestic and family problems and an inability on the part of the patient to adapt himself to the necessarily prolonged hospitalisation and all that it implies. In prevention of irregular discharges the supreme importance of a good doctor-patient relationship from the moment of diagnosis to final arrest emerges clearly, but, however good this may be, it may fail if unsupported by the essential ancillary services of nurse, social worker, rehabilitation expert, etc. There is undoubtedly great need for development and extension of these services. To attempt to treat only the local physical manifestations of the disease in the lungs and ignore its widespread effects on the victim as a whole can never solve the tuberculosis problem, as all physicians would agree. The evidence here presented serves to illustrate how far short of the ideal our present efforts fall, and indicates the directions in which considerable improvement could be achieved within the existing framework. F. H. S.

Report of the Ministry of Health for the year ending March 31, 1947.

Tuberculosis. Both the total number of deaths from tuberculosis (22,847) and the incidence as shown by notification figures (51,289) show a slight decline over the previous year. The disease accounted for 4·6 per cent. of all deaths. The number of institutional beds empty because of staff shortage increased to more than 15 per cent. of the available 32,000, while the number awaiting admission remained around 7,000.

The 18 Mass Miniature Radiography Units in England and Wales had examined 1,369,011 persons, and of these, 95 per cent. were reported as normal. Unsuspected active pulmonary tuberculosis was revealed in 4 per thousand and inactive P.T. (excluding calcified primary complexes) in 2 per cent.

In July 1946 the Ministry of Health agreed that B.C.G. production in this country should be explored and that subsequent trials might be planned. On the advice of the Medical Research Council authorised centres for the streptomycin treatment of tuberculous meningitis and acute miliary tuberculosis were organised in different parts of the country and were being extended as the drug became available. F. H. S.

NOTICES

NATIONAL ASSOCIATION FOR THE PREVENTION OF TUBERCULOSIS

NAPT Conference, 1949.

The Second Commonwealth and Empire Health and Tuberculosis Conference of the NAPT will be held in the Central Hall, London, on July 5, 6, 7 and 8, 1949.

The Minister of Health has sanctioned the payment of delegates' expenses upon terms of which the following is a brief summary. Further details may be had from the NAPT.

(*a*) Local Health Authorities in England and Wales:

The Minister has sanctioned the payment of expenses of one delegate for the full period of the conference, or two delegates for two days each.

(*b*) Regional Hospital Boards in England and Wales:

The Minister is communicating with the Boards and Hospital Management Committees on the basis on which he is prepared to approve payment of expenses in attending the conference, including conference fees.

(*c*) Local Health Authorities in Scotland:

The Secretary of State approves the payment of expenses of delegates within the limits prescribed by the Local Government (Scotland) (Conference) regulations, 1948.

(*d*) Scottish Regional Hospital Boards:

The Secretary of State will permit the Regional Hospital Boards to send delegates and to pay their expenses.

Provisional programmes will be sent on request. An extensive exhibition is being prepared in connection with the conference and also a series of visits. Fee for single tickets for four days of the conference, exhibition, etc., is three guineas.

NAPT Colonial Scholarships for Training in Tuberculosis in the United Kingdom, 1949

1. Owing to the success of previous awards, the NAPT has decided to offer six further Scholarships for postgraduate study in Tuberculosis in this country during 1949. They will be open to doctors and other medical personnel of either sex throughout the British Colonial Empire and the Sudan. The successful candidates will come to Britain for about six months to study Tuberculosis in its widest aspects: preventive, clinical, administrative and social.

2. Since Medical Departments may find it difficult to spare doctors, owing to the present staff shortages, the Council of the Association has decided to divide the awards as follows:

(*a*) Two Scholarships to doctors who either possess qualifications registrable in the United Kingdom (value of Scholarships, £120 each), or are graduates of Colonial Medical Schools (value of Scholarships, £100 each).

(*b*) Four Scholarships (value £80 each) to Matrons, Nurses, Health Visitors, or members of Colonial Sanitary Departments.

3. The successful candidates will be granted allowances to cover lodging and subsistence expenses from Colonial Government funds, and the details of their training during the tenure of the Scholarship will be supervised by the NAPT. Travelling expenses, purchase of books and other incidental expenditure will fall to be met by scholars out of their Scholarship moneys.

4. Application, which should be made through Heads of Departments, will be forwarded to the Colonial Office, who will make recommendations to the Council of the NAPT. Candidates should submit applications in time for them to reach the Colonial Office by May 1, 1949. A Circular Despatch on the subject is being addressed to Colonial Governments by the Secretary of State for the Colonies.

5. The NAPT believes that these awards will serve to further health education in its broadest sense, and in particular the organisation of anti-tuberculosis services in Colonial territories; and that the knowledge gained by those men and women who are able to come to this country and take advantage of the best that it can offer in the way of postgraduate opportunities will, through them, take root and spread throughout the British Colonial Empire.